of the second pulmonary embolism, the occurrence of the first pulmonary embolism and normal venography of the lower extremities strongly suggests that renal vein thrombosis preceded anticoagulant therapy and its withdrawal. The occurrence of renal vein thrombosis in the presence of minimal glomerular pathologic findings calls into question the hypothesis that histological changes are important determinants of renal vein thrombosis. Other factors, including hypercoagulability and disordered fibrinolysis, may be important in the pathogenesis of renal vein thrombosis.

Summary

Renal vein thrombosis developed in a 27-year-old man with long-standing lipoid nephrosis. In this case recurrent pulmonary emboli suggested the diagnosis. Selective venography showed a thrombus projecting from the left renal vein into the inferior vena cava, and anticoagulant therapy was effective. Although the exact mechanism of this complication remains unclear, abnormalities of coagulation or fibrinolysis may be important since histologic abnormalities are minimal.

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Staphylococcal Pericarditis

Successful Nonsurgical Treatment With Indwelling Catheter and Antibiotic Drugs

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AN ELDERLY WOMAN with staphylococcal pericarditis was cured with antibiotic therapy and an indwelling pericardial catheter without surgical drainage. The successful outcome of this case raises the question of when surgical intervention needs to be done in this disease.

Report of a Case

A 74-year-old woman with IgG multiple myeloma of ten months' duration was admitted to hospital because of progressively severe generalized weakness and fever. She stated she did not have chest pain. She had received her fourth course of chemotherapy four weeks before admission. The only abnormalities noted on physical examination were a rectal temperature of 39.4°C and a decubitus ulcer in the sacral area. There was no pericardial rub. Laboratory studies on admission gave the following values: hematocrit, 22 percent; leukocyte count, 1,500 per cu mm with mild left shift; platelet count, 57,000 per cu mm. An electrocardiogram showed sinus rhythm at 86 per minute and ST segment elevations in leads I, II, aV_L, aV_F and V_2 through V_6. The ST segment elevations had appeared since a normal tracing that had been taken four weeks earlier. An x-ray film of the chest showed mild cardiomegaly.

The high fever in this leukopenic patient with multiple myeloma suggested infection and prompted the administration of penicillin, 1 million units intravenously every four hours. By the third hospital day, the patient was afebrile, and cultures of blood, urine and sputum specimens taken at admission were reported to be sterile. After seven days the intravenous therapy with penicillin was stopped and administration by mouth begun. On the ninth hospital day, purulent fluid draining from the sacral decubitus ulcer was cultured. On the 11th hospital day, the tempera-

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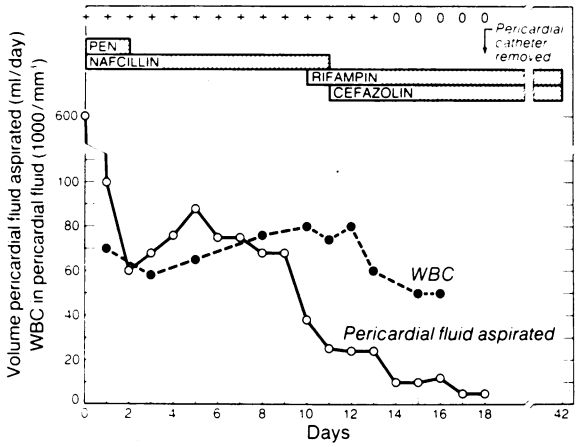

Figure 1.—Effect of pericardial drainage and antibiotic therapy on the volume and white blood cell (WBC) count of pericardial fluid: + = positive pericardial fluid cultures; 0 = negative pericardial fluid cultures.

ture, which had been normal for eight days, rose abruptly to 40°C, taken rectally. The patient looked extremely ill, but she had no chest pain.

On physical examination a regular pulse rate of 150 beats per minute was noted, with pulsus paradoxus of 10 mm of mercury, hypotension to 80 mm of mercury systolic pressure and pronounced jugular venous distention. An x-ray film of the chest showed a very large cardiac shadow. An M-mode echocardiogram indicated large anterior and posterior pericardial effusions and evidence of cardiac tamponade. Emergency pericardiocentesis yielding 600 ml of granular, purulent fluid was done using an 18-gauge Becton-Dickinson SH 2540 pericardiocentesis needle with a matched fenestrated Teflon catheter. The Teflon catheter, with a special plastic trocar to prevent clogging of the catheter lumen, was secured with its tip well inside the pericardial cavity for repeated aspirations of fluid. Gram stain of the fluid showed many neutrophils and many Gram-positive cocci, predominantly in clumps. Therapy with nafcillin sodium, 2 grams given intravenously every four hours, was initiated and the oral administration of penicillin was stopped. Intravenous administration of penicillin G was also resumed at the previous dose. Daily leukocyte counts and Gram stains and cultures of the pericardial fluid were carried out (Figure 1). Because pericardial fluid cultures grew *Staphylococcus aureus,* sensitive to nafcillin, cefazolin, rifampin and gentamicin but resistant to penicillin, the penicillin therapy was discontinued. The patient refused to have surgical drainage of the pericardium.

Despite the repeated aspiration of pericardial fluid and the intravenous administration of nafcillin, large amounts of purulent, culture-positive pericardial fluid continued to drain even ten days after the therapy was begun. Therefore, rifampin was added in a dosage of 300 mg given orally twice a day for its potential synergistic effects.[1] After 11 days of nafcillin therapy, a rash that was thought to be caused by this antibiotic appeared; the nafcillin therapy was then stopped and a regimen of cefazolin sodium (1 gram given intravenously every six hours) begun.

One day after rifampin therapy had been started, the amount of aspirated fluid began to diminish dramatically; after four days of rifampin (and three days of cefazolin) therapy, pericardial fluid cultures became sterile. After 19 days, the pericardial catheter was removed when fluid could no longer be aspirated and an echocardiogram showed no pericardial fluid. After having received a total of six weeks of antistaphylococcal therapy, the patient was discharged. When seen a year later, she felt well and had no evidence of infection and no cardiovascular abnormalities.

Discussion

This case report describes the successful treatment of purulent pericarditis with antibiotic drugs and the use of an indwelling Teflon pericardial catheter for repeated drainage of pericardial fluid. Several aspects of the clinical course deserve comment. Although electrocardiographic abnormalities compatible with pericarditis were seen at admission, the patient had no chest pain and no pericardial rub. The presence of chest pain in purulent pericarditis is unusual, having been reported in only 6 of 26 patients in one series.[2] The presence of a pericardial rub in this disease is also unusual.[2,3] In fact, the diagnosis of purulent pericarditis is easily missed because the classic symptoms and signs of pericarditis are often absent.[2]

In the past half century the spectrum of purulent pericarditis has changed with respect to the ages of the patients, the causative organisms and the factors predisposing to the development of the disease. In the preantibiotic era, patients with purulent pericarditis were generally young (mean age, 20 years), whereas now they are usually middle-aged and older (mean age, 49 years).[2,4] *Pneumococcus* was the most common infecting bacterium in the past, whereas now *Staphylococcus* and Gram-negative bacilli are the most frequently isolated organisms.[4,5] The main predisposing factors in acquiring purulent pericarditis before the availability of antibiotics were primary pulmonary

and pleural infections, most commonly pneumonia. Now, presumably because of advances in surgical techniques, antibiotic medications and chemotherapy, purulent pericarditis is seen most often in debilitated patients with impaired host defenses (such as those with carcinoma, uremia, hepatic failure, extensive burns, diabetes mellitus or leukemia and those patients receiving immunosuppressive therapy) and in otherwise healthy persons who have had extensive thoracic operations (such as an open-heart surgical procedure, penetrating thoracic injury repair and esophageal operation).[4]

Before the advent of antibiotics, surgical drainage was the only effective therapy for purulent pericarditis. Drainage resulted in about 50 percent survival in a disease that, without surgical intervention, had 100 percent mortality.[5,6] When surgical procedures could not be done, repeated pericardiocentesis was often used.[5,7] However, some authors argued that multiple aspirations of pericardial fluid could not cure the disease, and it was generally acknowledged that repeated pericardiocentesis increased the chances of serious complication. Interestingly, there were three reported cases (1921 and 1928) of patients with purulent pericarditis in whom closed pericardial drainage was done with an indwelling catheter.[8,9] In the two patients who recovered, the catheter was left in the pericardial space for periods of 28 to 41 days, without identifiable complications. Although superinfection did not occur in those two patients (or in the patient described here), it is clearly a risk of long-term catheter drainage.

Current opinion is that antimicrobial therapy alone is rarely effective in treating purulent pericarditis because pus and fluid reaccumulate, causing tamponade and creating an unfavorable milieu that decreases the effectiveness of phagocytes and antimicrobial agents.[2,5] However, a few reports describe the successful nonsurgical management of patients who were treated with antibiotic medications alone after an initial pericardiocentesis.[2,10,11] Some authors suggest surgical intervention only for those patients in whom recurrent tamponade, resistant organisms or spread of the infection occurs despite adequate and appropriate antibiotic therapy.[12] The concept that pericardiectomy may prevent or lessen the chances of a pericardial constriction developing continues to be debated. Opponents of that concept state that, in the absence of pericardiectomy, chronic constriction occurs only in patients with tuberculous pericarditis.[5,7] In our patient, surgical pericardiotomy was felt to be indicated because of persistently positive pericardial fluid cultures and reaccumulating fluid. However, as the fluid could be adequately drained by the indwelling catheter and as the patient refused an operation, surgical intervention was not done.

In this case rifampin was not used alone, for while in vitro studies have shown that many isolates of *S aureus* are sensitive to this antibiotic, clinical experience has indicated that resistance may emerge rapidly when it is the sole agent used.[13,14] Rather, rifampin was used as an adjunct to nafcillin (and later cefazolin) because the pericardial fluid continued to grow *S aureus* after ten days of treatment with appropriate antibiotic drugs. Clearly, it cannot be proved that in this case rifampin, as a bactericidal drug, was the major agent sterilizing the pericardial fluid because it is possible that continuing the administration of cefazolin alone might have achieved the same beneficial result.

Conclusion

Persistent pericardial drainage via an indwelling catheter and the administration of antimicrobial agents can cure patients with purulent pericarditis. The catheter may be left in the pericardial space several days for repeated or continuous aspiration of fluid, thus avoiding risk of complications from repeated pericardiocenteses. This treatment should be considered an acceptable alternative in patients with purulent pericarditis in whom surgical intervention appears to be extremely risky.

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